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Antiseptic cord care reduces bacterial colonisation but delays cord detachment

EDITOR,—We read with interest the article by Verber and Pagan on umbilical cord care.¹ As stated by the authors, although doubts have been cast on the need for antiseptic treatment of the cord,² the use of dry cord care alone leads to an unacceptably high colonisation rate of the umbilicus and exposes the neonate to the risk of infection.^{1,3} On the other hand, antiseptic treatment may delay cord separation, and no single agent has proved superior in preventing colonisation and disease.

We have recently conducted a prospective and controlled clinical study on 76 vaginally born healthy term neonates. Four different regimens for umbilical cord care were tested: ethanol 70% (n=19), chlorhexidine 0.5% in ethanol 70% (n=20), eosin 2% in ethanol 70% (n=18), and povidone-iodine 10% (n=19). The sample size for the study subgroups was established to detect a 50% difference in effectiveness between any two cord treatments, with $\alpha=0.05$, $\beta=0.20$. While in the maternity unit, the antiseptic agent was applied twice daily by a nurse; after discharge from the hospital, the same antiseptic preparation was applied 2-3 times daily by the parents, until cord detachment. An umbilical swab was taken from the base of the umbilicus on the first hour and the third day of life. All newborn infants were followed up for a one month period.

As shown in the figure, the percentage of positive cultures on the third day of life and the time to cord separation show statistically significant differences ($p<0.0001$) for the antiseptic agent applied. These results confirm that different antiseptic cord care regimens affect both bacterial colonisation rates and time to cord detachment, in an inverse relation. Chlorhexidine 0.5% showed the highest antimicrobial activity, with no colonisation by coagulase positive staphylococci nor group B streptococci; however, it was associated with

delayed cord separation. Ethanol 70% failed to prevent omphalitis, as all four cases detected during the study period occurred in newborn infants treated with this antiseptic agent. Eosin 2% and povidone-iodine 10% seem interesting alternatives; although these agents did not markedly reduce cord colonisation, both produced adequate detachment times, and no omphalitis occurred in these two groups. When pooled together, colonised cords detached earlier than those not colonised (mean (SEM) 8 (0.5) days *v* 14 (1) days; $p<0.0001$), irrespective of the antiseptic regimen. These data support the hypothesis that the use of antiseptic agents for umbilical cord care reduces bacterial colonisation and may inhibit leucocyte infiltration, thus delaying the separation of the cord stump.⁴

In our opinion, dry cord care is unacceptable, and the use of effective agents in cord care policies is highly desirable, if not mandatory. However, we are aware that it prolongs the interval between birth and umbilical cord separation. This fact may produce parental concern, increase the workload of community midwives, and prolong exposure to the risk of infection. Therefore, further research to shorten the time of cord separation, while applying appropriate antiseptic care, is needed.

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Selective intubation in a case of cystic adenomatoid malformation

EDITOR,—Although some cases of cystic adenomatoid malformation (CAM) may present in infancy or even later, the onset of clinical symptoms may occur in the neonatal period with signs of progressive respiratory distress related to the degree of insufflation of the cysts and the pressure exerted by the affected lung on healthy ipsilateral lobes and the contralateral lung.^{1,2} In this situation, reduction of cystic insufflation and stabilisation of the patient may be of great value before surgery.²

A case of CAM in which this was successfully accomplished by selective intubation of the contralateral main bronchus is presented.

Case report

A 21 day old infant was admitted because of respiratory difficulty of 24 hours' duration. On physical examination the patient was afebrile, with cyanosis and tachypnoea (100 breaths/min); there was hypoventilation of both hemithoraces with displacement of cardiac tones to the right. Chest radiography showed a huge air filled cystic mass with multiple septa in the left hemithorax displacing the mediastinal structures to the right and causing partial collapse of the right lung (fig 1). Arterial oxygen tension was 3.3 kPa, carbon dioxide tension 12.9 kPa, pH 7.07, and base

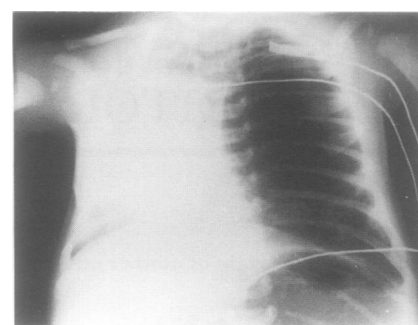


Figure 1 Initial chest radiograph.

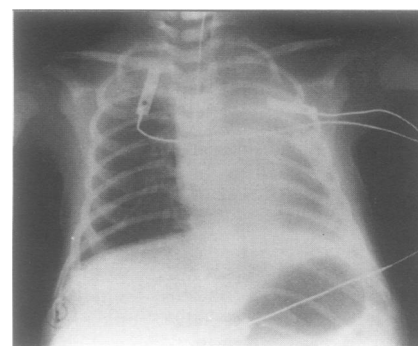


Figure 2 Chest radiograph after selective intubation of the right bronchus.

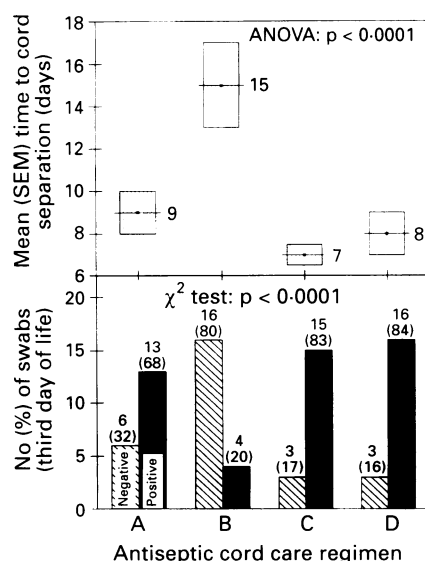
excess 2 mmol/l, and failed to improve after conventional intubation and mechanical ventilation. In view of the patient's clinical status, selective intubation of the right bronchus was done, with clinical and gasometric improvement. On repeated chest radiographs the right lung appeared normal, the mediastinal structures had returned to the midline and the degree of insufflation of the left pulmonary cystic lesion had become strikingly smaller (fig 2). Computed tomography of the chest revealed images suggestive of CAM involving the left lower lobe. The patient underwent left lower lobectomy. The postoperative course was uneventful and assisted ventilation was withdrawn 72 hours after surgery. Pathological study confirmed CAM type I. Twenty days after admission, physical examination was normal and the patient was discharged.

The clinical pathogenesis of CAM derives from the progressive distension of the cysts that may lead to increased intrathoracic tension, compression of the healthy ipsilateral lung and the diaphragm, displacement of the mediastinum, and atelectasis of the contralateral lung.^{2,3} In cases in which this situation provokes severe respiratory embarrassment, decompression of the cysts before surgery may help to improve ventilation.

We have successfully used selective intubation of the contralateral bronchus in cases of acquired bronchopleural fistula. This procedure is generally well tolerated.⁴

In the present case using selective bronchial intubation, we reduced the degree of insufflation of the cystic malformation and in consequence improved ventilation of the contralateral lung. With this manoeuvre, the patient remained clinically stable for the 24 hours before surgery with stable blood gases.

A review of the literature yielded no other case of selective intubation of the healthy lung in patients with CAM. We therefore consider our case to be of interest as this procedure may be effective in improving the condition of the patient before surgery.



Time to cord separation and number of positive and negative swabs on the four regimens. A=ethanol 70%, B=chlorhexidine 0.5%, C=eosin 2%, and D=povidone-iodine 10%.

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Diagnosis of non-immune hydrops in the newborn

EDITOR,—Stephenson *et al* provide a helpful 'personal practice' article in relation to non-immune hydrops of the newborn and a near comprehensive list of reported associations.¹ With such heterogeneity in the causes and associations of non-immune hydrops it is perhaps inevitable that the list is not absolutely complete and I write to highlight one potentially important omission, namely congenital myotonic dystrophy (CMD). Stratton and Patterson recently confirmed this diagnosis by DNA mutation analysis in a case of non-immune hydrops and provide a good literature review of hydropic infants born to mothers with myotonic dystrophy, a total of 16 cases including their own.² Such numbers, although only a minority of those with CMD, suggest a clear cause and effect relationship rather than a chance association. The precise pathophysiology is undetermined but myotonic dystrophy is a multisystem disorder with cardiac muscle and conduction pathways significantly affected in a proportion of patients. There is good evidence that earlier onset, more severe forms of myotonic dystrophy will fair worse in this respect. The

youngest case personally known to me who required a pacemaker did so at 16 years of age. It is reasonable to postulate that non-immune hydrops in CMD may result from unusually severe cardiac muscle involvement with intrauterine heart failure – but this is speculation at present. As Stratton and Patterson point out,² in unexplained non-immune hydrops an examination of the mother and a detailed family history may provide the all important clues. The inheritance of myotonic dystrophy is autosomal dominant and the phenomenon of 'anticipation' is observed, that is, the age of onset of symptoms is earlier, and the severity of most features increased, with succeeding generations. It is virtually always the case that the affected parent of a baby with CMD is the mother and she can be expected to show at least some clinical signs of the disorder. However, the diagnosis must be considered first. Once CMD has occurred in one pregnancy it is highly likely that the next fetus to inherit the myotonic dystrophy gene will also manifest the congenital form. Of course, once a diagnosis of CMD/myotonic dystrophy has been made then genetic counselling and testing can be offered to other family members at risk.

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Patent ductus arteriosus in the newborn

EDITOR,—In situations and places where injectable indomethacin is unavailable, oral indomethacin has been used for closure of the patent ductus arteriosus (PDA). It is, however, extremely difficult to fractionate accurately 25 mg of the capsule (powder) into 0.2 mg sachets or doses, especially when sensitive weighing scales may also not be available. We noted that oral indomethacin powder does not disperse easily in water or other liquid vehicles. We have hence used

mefenamic acid, another non-steroidal anti-inflammatory drug, as an alternative for treatment of symptomatic PDA.^{1,2} Mefenamic acid (Ponstan, Parke-Davis 50 mg/5 ml) was administered orally in three doses of 2 mg/kg/dose at 12 hourly intervals in 25 neonates with symptomatic PDA.³ The mean gestation age of these neonates was 30 weeks, mean birth weight 1320 g, and mean age of administration of mefenamic acid was 16 days. Twenty neonates had pretherapeutic two dimensional echocardiography and Doppler flow studies performed to confirm the diagnosis of PDA and to rule out a ductus dependent state. Twenty two patients (88%) clinically responded to this therapeutic regimen within 48 hours of administration of the last dose of mefenamic acid. Of three non-responders one was subsequently diagnosed to have an endocardial cushion defect and the other two were then administered three doses oral indomethacin (0.25 mg/kg dose) with no therapeutic response.

Our earlier experience with the use of oral indomethacin in 32 preterms of mean gestational age 31 weeks indicated a PDA closure rate clinically of 75% in those treated with indomethacin.

Our initial experience with mefenamic acid treatment, although restricted to 25 cases, suggests that it is effective, safe, well tolerated, and certainly easier to administer than oral indomethacin. In situations where injectable indomethacin is not available, we suggest that mefenamic acid be used given its efficacy and ease of administration of accurate dosage. Further trials need to be conducted in larger number of cases to confirm our observations.

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